ORIGINAL TRANSCRIPT

U. S. ENVIRONMENTAL PROTECTION AGENCY

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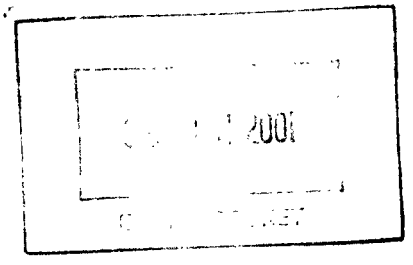
EPA SCIENCE ADVISORY BOARD
CLEAN AIR SCIENTIFIC ADVISORY COMMITTEE
CASAC PARTICULATE MATTER REVIEW PANEL

MEETING



July 23, 2001

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EPA SCIENCE ADVISORY BOARD

CLEAN AIR SCIENTIFIC ADVISORY COMMITTEE (CASAC)

CASAC PARTICULATE MATTER REVIEW PANEL

July 23, 2001

DR. HOPKE: Let me get this show on the road here. Good morning, ladies and gentlemen. I am Phil Hopke. I am the chair of the Clean Air Scientific Advisory Committee, and we are here today to review the draft Criteria Document on particulate matter, and then, tomorrow, we will also be taking a look at the very preliminary version of the staff paper and, particularly, the approaches to be taken with regard to risk assessment and urban visibility assessment.

As you are aware, this is a public meeting being held under the FACA rules. What we have done, in order to try and expedite the process, is to have written statements by the panel members describing some of their background and related information which we have often, in the past, described orally and which we are going to bypass today, in general, because we have got the written

material out there on the table for any of you who wish to obtain that.

So, first, what I would like to do is go around the table and have people introduce themselves, and then we will come back to the one bit of background that we do need to pick up orally. So, why don't we start with Warren and come around the table?

DR. WHITE: Warren White, Washington University in St. Louis.

SPEAKER: I would like to remind the panel members to use the microphone whenever possible to make sure their voices carry in the room. You have to use the larger microphones.

DR. WHITE: Warren White, Washington University in St. Louis.

DR. MAUDERLY: Joe Mauderly, Lovelace Respiratory Research Institute in Albuquerque.

DR. SAMET: Jon Samet, Johns Hopkins School of Public Health.

DR. UPTON: Art Upton, University of Medicine and Dentistry New Jersey, Robert Wood Johnson Medical School.

DR. HOPKE: I am Phil Hopke from Clarkson University.

MR. FLAAK: I am Bob Flaak from EPA's Science Advisory Board staff.

DR. MILLER: Fred Miller with CIIT Centers for Health Research here in the Park.

DR. MCCLELLAN: Roger McClellan, private advisor, Albuquerque, New Mexico.

DR. KOUTRAKIS: Petros Koutrakis, Harvard University.

DR. LEGGE: My name is Allan Legge with Biosphere Solutions in Calgary, Alberta in Canada.

DR. TAYLOR: George Taylor with George Mason University in Fairfax, Virginia.

DR. ROWE: Bob Rowe, Stratus Consulting in Boulder, Colorado.

DR. KOENIG: Jane Koenig, University of Washington, Seattle.

MR. FLAAK: Thank you, everybody.

The folks in the back of the room, can you hear okay, or is it a little bit difficult? Microphones are up loud enough?

Okay. For the purpose of the folks at the table, the smaller microphones are for the court reporters, so if you speak loudly into those, you'll see these two gentlemen over here jump about five feet, so try and use the big mikes.

Let me just cover a couple of administrative things for today. We have a very busy agenda. There is an agenda available for everybody. I suspect most of you have picked it up by now. If not, it is outside on the table. It covers the general discussions at the meeting.

Just as a reminder, what we are doing here today is the committee is conducting a peer review of the draft Criteria Document for particulate matter, and there are preliminary comments from the committee members which are included on the table outside. If any of you did not get a copy of those preliminary comments because there were insufficient copies available, it is on our web site as of today, and you can get a copy of it from there as well. If you need the web site address, it is www.epa.gov/sab for Science Advisory Board.

If you need that information again later, just check with me.

The disclosures that we typically do at our meeting, as Dr. Hopke mentioned, have been handled in writing at this meeting, so there should be copies of that available to all of you to get a sense of the background of all of the committee members. One of the statements I didn't get in time to include in here, and that is from Dr. Koutrakis, and I will ask him in a moment to do his orally just to get it on the record.

There is a dinner scheduled for this evening for the committee. It is going to be at the Aurora Restaurant which is about seven miles down Route 54 toward Durham, and I would like to get a hand count, so I can call the restaurant, of how many people to expect. How many people will be joining us this evening? And everyone is welcome to come.

(Show of hands.)

MR. FLAAK: All right, thank you.

At this time, I would like to...yes?

DR. GRANT: The restaurant is

actually in Chapel Hill.

MR. FLAAK: Say it again?

DR. GRANT: Chapel Hill, Route 54.

MR. FLAAK: Route 54.

DR. GRANT: Route 54 just on the edge of Chapel Hill.

MR. FLAAK: Thank you. I would also like to introduce Ms. Rhonda Fortson. Rhonda, if you would, stand up for a second. Rhonda is our new staff member on the Science Advisory Board. Thanks. She will be supporting CASAC in the future. She has joined us from the EPA Athens Laboratory. So, for those of you around the table who will be doing travel and other things with us, Rhonda will be the person you will be talking to. She will be with us for part of today. If you have any questions about your travel, please check with her. And you will be leaving, what, about 2:00 o'clock this afternoon to head back to Washington.

Petros, can I ask you to give a brief disclosure statement?

DR. KOUTRAKIS: My name is

Petros Koutrakis. I am a professor of environmental sciences at the Harvard School of Public Health. My research includes exposures and health effects of ambient particles. I have funds from EPA and other sources, and I have made public statements in hearings and interviews about the effects of ambient particles.

Is that enough?

MR. FLAAK: I think so.

One of the other topics the committee will be taking up today is a consultation on the staff paper, the preliminary version of the staff paper. The difference between a peer review and a consultation, for all of you that may not be familiar with it, in a peer review, we actually do a full review of the document, provide comments, and produce a report which goes to the Administrator afterwards. In the case of the Criteria Document, that report should be ready approximately 30 to 60 days following this meeting.

In terms of a consultation, a consultation is an early discussion that the Science Advisory Board holds with the program

staff on a topic. Often, it is early in its stages of preparation before a lot of additional changes need to be made, and in the case of the staff paper, that is what we will be doing, and that, actually, will take place after lunch tomorrow.

There will not be a written report from the committee. We are not seeking consensus views. We will be having an open discussion on the staff paper. There are some individuals who have provided individual comments on that document, and those are also included on the table outside, and the only way that the Agency will get advice from the committee on that document will be through those individual comments.

As I said, this is not a closure issue. We will be reviewing that document at a later date and, probably, Karen will give us some sense of when that might be.

Does anybody have any questions about the process we are going to follow today?

(No response.)

MR. FLAAK: Okay. The agenda is pretty busy. We have a lot of speakers

that are scheduled for today. I will introduce the public comment period at approximately 10:00 o'clock, and I will give you some instructions about how we are going to do that.

Karen?

MS. MARTIN: Yes, I just wanted to offer some clarifying comments or explanation with regard to the status of the particulate matter standards review in light of the ongoing litigation from the standards promulgated in 1997. There are a lot of questions as to how those things play out together, and I wanted to just clarify what the situation was at this time.

As I am sure you know, the litigation has been ongoing for the '97 standards. You are probably aware that the Supreme Court issued a decision earlier this year upholding the constitutionality of the Act and our interpretation of it and, also, reaffirming that we are not to consider cost in decisions on the standard.

That did not, however, of course, end the litigation, and I wanted to make clear

that what it did was send the case back to the D.C. Circuit Court of Appeals to consider remaining issues that the Court didn't address when they first had the case before their initial decision, and the Court has now set a briefing schedule for their further deliberations which extends through November of this year before final briefs are due to the Court.

That schedule implies, of course, that it is unlikely that we hear a decision back from the Court on those standards until next year, and when next year is a matter of speculation that I won't speculate on, but we are very unlikely to hear back before the beginning of next year.

The ongoing litigation on the NAAQS standards, I only want to make the point that it doesn't interfere with our current review. The initial decisions that did come out of the Court, they did not revoke the fine particle standards, the $PM_{2.5}$ standards, so they remain in place although continued subject of litigation.

They did, however, revoke the revised

PM_{10} standards which were put in place to address the coarse fraction particles on the basis that PM_{10} and fine were overlapping indicators, and that left in place the 1987 PM_{10} standards so that what we have in place right now are 1987 PM_{10} standards, 1997 $PM_{2.5}$ standards, and the upshot of that is, of course, it places particular attention in this review on considering the coarse fraction particles and indicator for coarse fraction particles and then, from there, consideration of other elements of the standard.

With regard to where we are going from here in the staff paper, Bob stressed the point that this was early in the process and we are seeking early consultation. What has become clear to us is that when we put out the Federal Register notice last month releasing this preliminary draft staff paper, we weren't clear enough with regard to what additional next steps would be happening that would provide further opportunity for public comment.

In point of fact, the risk assessment will be presented in more detail in a

subsequent document which will come to this committee sometime this fall, laying out a more complete methodology for risk assessment, and that document will be informed by the consultation we are having with you as well as public comments on the initial scoping. So, there will be a second opportunity for comment prior to actually conducting a risk assessment and presenting results in the next draft of the staff paper.

Likewise, with the urban visibility discussion, the consultation now is to get early input on analyses. There will be a Federal Register notice laying out that in more detail prior to commencing any further work in that area as well.

So, I wanted to make the point that, in both of those cases, there would be a second pass for public comment and comment from this committee prior to actually conducting analyses and incorporating them in the next draft of the staff paper.

The next draft of the staff paper will, of course, fully recognize changes being made to the Criteria Document in light of this

review in the next couple days and comments, and that suggests, then, of course, we are not going to see another draft of the staff paper until next year as well.

I would just end by making the point that in order to help clarify the early stages of the staff paper and these additional opportunities for public comment, we will be...we have prepared and, in the next several days, will be releasing a Federal Register notice explicitly extending the public comment period on the preliminary draft through the end of September and laying out the other documents that will be following so that there is a clear understanding of what future opportunities there are for input.

DR. MCCLELLAN: I am still not real clear. Maybe you could elaborate a bit. You are extending the public comment period on the staff paper, and then you are coming back to the CASAC PM panel with an updated risk analysis plan, and that would be what time period?

MS. MARTIN: I would hope within the next couple months with a more

detailed risk assessment methodology plan.

DR. MCCLELLAN: Okay.

MS. MARTIN: To get additional input prior to conducting the assessments and incorporating results in the next draft of the staff paper.

DR. MCCLELLAN: So, that might be available, say, first of October perhaps?

MS. MARTIN: Perhaps around there, yes. I don't...

DR. MCCLELLAN: And that might mean, then, a CASAC PM panel meeting maybe in December?

MS. MARTIN: Whether it is conducted in conjunction with a meeting or whether it is written commentary, we have yet to discuss exactly the best way to go with that, and perhaps hearing comments tomorrow afternoon will provide us a better indication of what the most appropriate method would be.

DR. MCCLELLAN: So, that means, then, you would begin work on the risk assessment in, perhaps, January or the beginning of 2002?

MS. MARTIN: Late this year, if

at all possible.

DR. MCCLELLAN: And then, how long do you anticipate that will take?

MS. MARTIN: That won't necessarily take a long period of time, depending on where we go with the assessment. So, that is yet to be determined, even the scope of the assessment, so it is a little hard to predict a length of time it will take to complete it.

DR. MCCLELLAN: Okay.

DR. HOPKE: You are going to look for us to provide a formal review of the risk assessment plan?

MS. MARTIN: We are going to look for at least an additional consultation on a more detailed methodology document.

DR. HOPKE: All right. I would guess that we would probably plan to try and do that through a teleconference.

DR. MCCLELLAN: Here is what I was suggesting, Phil. It seems to me that risk assessment plan, this is a consultation on it, but I would urge that the Agency give the public ample opportunity to comment on that

risk assessment plan, and that may well include not just written comments but the opportunity to offer oral comments on it, and it would seem to me to be imperative that the panel offer a written set of comments back in the way of guidance to the Agency on it.

So, I was just trying to get a feeling for this time period on it. As much as we would like for it all to move fast, it seems to me that the actual facts in terms of the need to have time to do a quality job are going to mean this thing is going to take more time, probably, rather than less.

DR. HOPKE: Other questions for Karen?

(No response.)

DR. HOPKE: Okay, good. Well, we are moving a little ahead of time here.

SPEAKER: Won't last.

DR. HOPKE: Won't last, right. So, let's turn things over to Dr. Grant who will then provide us with an overview of the air quality criteria for particulate matter from the second external review draft. Les?

DR. GRANT: Okay. Well, good

morning. It is nice to be back with CASAC again, Phil. We have a couple interesting days ahead of us, I guess.

Where to start? Dennis, if you would like to turn on the first slide up there, I think it might be useful for us to just go back over a bit in the way of the time line and some key milestones in the development of the document here that you have before you.

I should note at the outset...next slide there, Dennis...if you recall, back in 1997, at the time that the final decision was promulgated, there was an issue, the Presidential memorandum, that basically indicated that we, EPA, should specify a schedule for completion of the next round of Congressionally mandated review of the standards, and that meant also publishing, in October of '97, the schedule in order to, ideally, reach a complete round of review, preparation and review of the Criteria Document, staff paper, promulgation...or, excuse me...the publication of proposed retention or revision of the standards, and then a decision, as Karen mentioned, by July of 2002.

We did, rather immediately, start the development of the Criteria Document and, as indicated up here, had the development plan reviewed by CASAC at a May, 1998 meeting. We went along through, then, to prepare the first external review draft of the document, basically, according, as close as possible, to that originally stated schedule. That document, dated October of '99, was reviewed at a CASAC meeting in December of '99. When I say reviewed, it was actually a consultation.

The reason it was a consultation was that it was recognized that there was a tremendous amount of new research information that would be coming out through the course of the next six, seven, eight months or a year or whatever, that needed to be incorporated into the second external review draft.

There are several things that occurred, then, to help facilitate bringing out that new information. One of the major events was the PM 2000 International Conference that co-sponsored by EPA and a number of other groups and held by the Air and Waste Management Association in January of 2000. That provided

an opportunity for presentation, both platform and poster session-type presentation, of new research results from EPA and outside of EPA, the general scientific community in the United States and internationally.

We arranged for expedited peer review of PM 2000 journal articles that the authors and so on chose to go ahead and undertake with us. Many of those took a bit longer than we thought or had hoped to be able to get them out into the journals. They really began appearing August through December.

There were a number of key HEI reports, things such as the NMMAPS and 6-Cities and ACS reanalyses and so forth, that came out through the June to December period. So, we struggled through last fall to try to incorporate as much of the new research into the document in an effort to bring it out by the end of the year.

Though it took us a bit longer than that, we finally were able to go ahead and wrap up and put out a draft dated March, 2001 and started a public comment period April 12th through July 12th, leading to this CASAC review

meeting.

A number of folks have asked well, gee, what does that mean as far as what happens next? Is this going to be it, the final document or whatever? And we have been very straightforward, in fact, have stated in some various public meetings and so on that that was quite an unrealistic expectation.

We fully expect to put out a third external review draft, taking into account the public comments received on this one and, also, the review and comments from CASAC, from this committee. Our target, in general, is to try to produce that next external review draft, if possible, by the end of this year in time for a public comment period running early next year and CASAC review early next spring.

Hopefully, then, three to four months later after that, if we are able to achieve closure at the next meeting, CASAC review on that third draft, three to four months after that to try to produce the final document. That is sort of, roughly, what we expect or hope.

I think it is useful, Dennis, to go

ahead and put the next slide up and to just note this new document has really been quite a challenge for us. There are massive amounts of new information that are now considered in this second external review draft. There are about 1800 new references, is our estimate, that we are citing in here since the October, 1999 first review draft was out.

This reflects outputs from tremendously expanded research programs both within USEPA, both the intramural and extramural programs, as well as numerous other Federal and State agencies. The Health Effects Institute is one of the key non-governmental groups with very major research efforts going on on PM, and then, quite a number of other research organizations both here in the U.S. and internationally.

I think this reflects quite intensive efforts by the researchers in the general scientific community and a lot of cooperation on their part to try to produce and publish the outputs from this research in a timely manner, and we do appreciate that.

I think we also do owe a special note

of thanks to some of the organizers and co-sponsors of some of the various meetings that helped facilitate public vetting of the new research findings. They include the Third Colloquium on PM Air Pollution and Health that was held here in Durham last June, if I recall correctly...not this June but the prior June of 2000; the AWMA PM 2000 Conference that I just mentioned; also, I think, the HEI Meeting on Fine Particles along with the European Communities.

We also, I think, owe quite a debt of appreciation to the journal editors that helped expedite peer review and publication of PM papers. Those include the editors for Aerosol Science and Technology, Journal of Air and Waste Management Association, Journal of Exposure Analysis and Environmental Epidemiology, Inhalation Toxicology, and Environmental Health Perspectives. There are some other ones, but these are key ones, and we really do appreciate the efforts on the part of folks, including some of our CASAC members here such as Phil Hopke and Petros Koutrakis and so on. Ito Pelasari is not

here, but...and Don Garner as well for some of the other journals.

In any case, production of this second external review draft included quite an extensive effort, not only by myself but quite a number of other members of our scientific staff in the National Center for Environmental Assessment in RTP. I just want to take one moment. The names of these people are listed in the front matter for the Criteria Document. I am joined here at the table today by William Wilson to my right and also by Allan Marcus, then, to his right as two key people who are going to be presenting some further information as part of this overview in a few minutes.

I should also add recognition for Lawrence Follensbee, Larry Follensbee on my staff, Jay Garner, Dennis Kotchmeyer, Robert Kaplan, Beverly Comfort, William Niemald, David Mage who has now gone off to Temple University, Allan Marcus I did mention, Jim McGrath who is a visiting scientist with us and then has gone back to Texas Tech University, and Joseph Pitthou and James Robb as having, on my staff, provided quite key

inputs in production of the document.

There are also numerous other consultants that have worked with us in helping to author sections and other reviewers. They are too numerous to recognize here. They are listed in the front matter of the Criteria Document, and we do send our appreciation to them. Some of them are here with us to help in the course of today's discussions.

Moving on, Dennis, to the next slide, what we have in mind is to just run through very quickly a few things on each of the different chapters, a little bit of highlights or notes, and to highlight, perhaps, some key issues from these and some examples of new research that we expect to bring into play of some of the numerous research studies that are now appearing or starting to appear and have appeared since we closed up the document.

I should note with regard to that, we tried to be as inclusive as we could of the most important and pertinent information that was available through December of last year. We didn't get all of that information. It was really tough, believe me, in terms of the flow

of the information and the amount of papers coming to just keep up with all of it. So, we are not entirely inclusive all the way through December.

Since December, there are quite a number of additional papers that have come out that we have been trying to collect and get into the hands of our staff members and consulting authors to already be working to start summarizing that new material and looking at it. We expect to hear still more in the course of these discussions, and I noticed in public comments that we have looked over, additional studies being identified.

So, our intent, in terms of the production of the next external review draft, is to incorporate any newly available studies up through, essentially, this month, July of 2001, anything that has been peer reviewed and published or accepted for publication, basically, through the end of July here as a cutoff point.

Obviously, if there is some truly momentous whatever, new paper that comes out that is of such monumental importance, such a

significant increment in information or knowledge or bearing on crucial conclusions or whatever that may appear after this, we will consider that and probably consult with the committee with regard to whether, you know, to incorporate any such, shall we say, notable new contribution that is of such importance, you know, as to violate our cutoff date here as the end of July.

Anyway, so end of July as a cutoff point for information going into the next drafts of the document.

Turning, now, to a very quick overview of the document with regard to what is in the different chapters, the first chapter, the introduction, basically provides important background information for the rest of the document. It does talk about the legislative requirements, provides a history of the previous PM Criteria and NAAQS reviews, talks about the current PM Criteria and NAAQS reviews, and it has information on document content and organization.

The next slide, I am going to turn it over, then, to William Wilson to talk about

Chapters 2 through 5 and then, afterwards to have Allan Marcus to cover Chapter 6, and then back to me to cover the last few chapters.

William?

DR. WILSON: Chapter 2 covers the physics, chemistry, and measurement of particulate matter. Compared to the previous Criteria Document, we have more emphasis on the properties of ultrafine particles which some people call the nuclei mode and some people call nanoparticles as well as being called ultrafine. We are also addressing more thoroughly the problems of measuring semivolatile aerosol components.

I want to emphasize that Chapter 2 does not address issues related to NAAQS implementation. It only addresses those issues relevant to reviewing the science pertinent to the NAAQS standard setting.

This is partly due to requests from CASAC and others that we reduce the volume of the Criteria Document. We have also chosen to do this because much of this material on air quality modeling, aerosol equilibrium models, and other topics that are related to

implementation are being addressed in the NARSTO Science Assessment documents which are being prepared right now, and NARSTO is the North American Research...anyway, it is a group that includes the U.S., Canada, and Mexico and originally started out with emphasis on ozone. I guess that S-T-O is the Science of Tropospheric Ozone. They have added particles to what they are concerned with and will have an assessment of the state of science as it applies to implementation.

There are a number of new papers which are relevant to the Federal Reference Method, and I won't go over them, but you can just put up the next slide, too, Dennis. We will include these in the review, because they provide some information on how well the new measurement method works and some intercomparison studies. So, that will be an important addition.

I would also mention that we talk a little bit in Chapter 2 about the analytical techniques that are needed to do source apportionment modeling, and since epidemiology and toxicology are beginning to use source

apportionment as a tool, we have included some information on source apportionment modeling in Chapter 3.

Go ahead, Dennis. In Chapter 3, the current version has the 1999 annual mean concentrations for $PM_{2.5}$, PM_{10}, and $PM_{10-2.5}$. We will be adding the 2000 data, but we need to emphasize that we need three years before we can determine attainment status.

Go through the next two, Dennis. We have shown in handouts the $PM_{2.5}$ and $PM_{10-2.5}$ concentrations.

Chapter 4 is Environmental Effects of Particulate Matter: Effects on Vegetation and Ecosystems. I am going to go through this fairly quickly because of the time constraints.

We have made effort to cover much of the chemistry and physics related to the biochemical cycling, and we have made use of a number of other assessment and extensive studies carried out either in this agency or other agencies that relate to this. One is the Integrated Forest Study which includes some of the things like nitrogen and sulfur deposition.

Next slide, Dennis. The National Acid Precipitation Assessment Program State of Science Report is a source document which we refer to and includes much of the information relevant to acid deposition.

Next slide. We have also looked at the effects of particulate matter in reducing light penetrating the surface vegetation, because this may have effects on the yields of as well as production due to pollutants reacting with the light.

We also reviewed the status of information on visibility effects of particles...next...and what information is available on the effects of particles on materials.

The effects of particles on climate change processes and the potential human and environmental impacts are a subject of great concern right now, and there are a number of comprehensive assessments by other government, both national and international agencies and groups, so we refer to those in the Criteria Document.

We are, of course, interested in

CASAC's comments on what should be added or emphasized or changed in this chapter.

The next chapter is Human Exposure to Particulate Matter and Its Constituents. We have highlighted a number of the issues that are discussed in the Criteria Document. I don't think I need to go through these in detail. We find that there is really not a great deal of information and experimental data to address many of the issues that we are concerned with.

So, if you will, just go through, Dennis, to the one that shows the new papers. There are a number of new papers which have been accepted for publication now and which will provide some very important new information, and I think it may be the next slide or so where we have the new papers, Dennis, and we'll finish up with that. Yes, here are examples of some of the new studies, and these include how you can use the extent of air conditioning in different cities to account for some of the variation in health effects found in different cities, some additional information on the role of gaseous

pollutants as cofounders, some new information on the toxicity of indoor-generated particles and how that compares with outdoor particles, and some new information on the relationships between indoor/outdoor and personal exposures from EPA's study in Fresno.

There are a few other studies that are listed, including EPA's study in Boston, a panel study in which both exposure parameters and health parameters were measured.

So, that concludes through Chapter 5. I would just say that we are looking forward to the comments of CASAC.

DR. GRANT: I just should note, in particular, back with regard to the climate change information that is in there, what we tried to do was to provide, especially in appendix materials but also in the main text, information drawn from a number of other rather extensive reports, as William noted, and several of those have been in the process of being drafted, and now, we probably have in our hands more recent draft material than what is reflected in the chapter right now or in the appendix of the chapter, which we would,

of course, want to update in the next review draft to reflect the latest final versions or whatever of some of these materials that are being produced both internationally and here in the United States with regard to climate change aspects.

DR. HOPKE: Okay. Let me take a brief pause here.

DR. GRANT: Sure.

DR. HOPKE: We have had a couple people join us, so if, Paul and Morton, you could introduce yourselves just briefly, your name and where you are from.

DR. LIOY: Good morning. I am Paul Lioy, member of the SAB and a member of the Clean Air Compliance Council and a consultant to CASAC. I am the associate director of the Environmental and Occupational Health Institute in New Jersey and professor of environmental and community medicine.

DR. LIPPMANN: I am Morton Lippmann, NYU, professor of environmental medicine at New York University School of Medicine and a member of this panel.

DR. HOPKE: Thank you. Are

there any clarifying questions for Dr. Wilson before we move on? We will have adequate time to discuss the chapters individually, but are they any quick questions anybody has before we move on?

(No response.)

DR. GRANT: Okay, thank you. Okay, Dr. Allan Marcus, biostatistician on my staff, will provide some comments with regard to the materials presented in Chapter 6 dealing with the epidemiology aspects.

Allan?

DR. MARCUS: Okay, let's see if we can get into the 21st century here as far as presentations are concerned. I am briefly going to review some of the material in the epidemiology chapter providing, basically, an enlarged table of contents.

The key endpoints that are evaluated in the chapter are, first of all, mortality, then hospital admissions for cardiovascular and respiratory causes, respiratory illnesses and symptoms, and physiological changes that appear to be precursors of adverse health effects.

The time scales for these effects are

several. Most attention has been paid to acute effects occurring hours or days after elevated air pollution exposure. There have been a number of important studies, however, on long-term effects occurring after months or years of exposure and a very few studies using new methods looking at effects occurring after a few weeks to months of air pollution exposure. I will call those semi-chronic.

The short-term particulate matter exposures from air pollution monitors often show significant positive associations with daily mortality and hospital admissions. The NMMAPS study is particularly important, because it includes the largest number of cities, 88 cities, all of them evaluated using a virtually common methodology, 88 cities in the contiguous 48 States, and this allows us some assessment of spatial heterogeneity, and it does appear that there is some heterogeneity in the effects across different regions of the U.S. These findings have not yet been confirmed or explained.

Associations between $PM_{2.5}$ exposure and daily mortality are stronger than

those...usually stronger than those between mortality and the coarse fraction, $PM_{10-2.5}$, but the amount of available data is very limited.

So far, there is one study available using particles in various ultrafine fractions in Erfurt, Germany, but it is, at this point, too soon to know whether or not the results are generalizable to ultrafine particle fractions in the U.S.

Statistically significant associations with daily mortality may be greatly reduced when the mass of PM_{10} is dominated by crustal particles, and this is beginning to show up in studies, for example, in Spokane and in some of the cities in the Utah Wasatch Front.

Limited data suggests that fine particle associations may be greatly reduced when crustal particles dominate the intermodal fraction of the fine particles as is suggested in a recent paper by Clayburn et al for Spokane.

Probably the biggest or one of the most important new developments gives us a bit of a handle on composition effects. This uses...most of these approaches use regression

analyses based on principal components or factors of speciated fine particles by elemental composition, and these suggest much higher associations of excess mortality in time-series studies with combustion-related PM components. The combustion-related components are motor vehicles, oil or coal burning, and, in some places, wood burning. There is also some indication of a regional sulfate effect associated with excess mortality.

These effects are, generally, much stronger than those associated with soil and crustal particles which are, generally, statistically not significant. However, the conclusions are based on a small number of U.S. cities and one Canadian city, they use a diverse set of statistical methods, and it would be nice to see considerably greater confirmation of these studies, particularly in western sites.

In spite of a great deal of very interesting and important new work in this area, there is still, certainly based on the comments I just received, the public comments I have just received over the last few weeks, a

great deal of interest in the co-pollutant issues. These are described in the Criteria Document mostly in terms of whether or not it is possible to assess independent effects of human health effects associated with the gaseous criteria pollutants, ozone, carbon monoxide, nitrogen dioxide, and sulfur dioxide. Also, there is very little information on the extent to which the gaseous criteria pollutants exacerbate or interact with health effects of airborne particles, even at low levels.

Among several methods that are available, one is use of meta-regressions adjusting for mean or median concentrations of gaseous co-pollutants. There are concerns, still, about the extent to which spatial measurement error among the differential spatial measurement error across the different pollutants might affect the robustness of the estimates of PM_{10} or fine particle effects on mortality and hospital admissions.

Finally, while the most conventional approach, namely, using multiple pollutants in the model simultaneously, that is, a PM index or indices plus one or more of the gases,

there is still considerable discussion about how to interpret these findings, and any CASAC recommendations as to how to deal with the alternative statistical interpretations or approaches for addressing the question of potential co-pollutant confounding or interaction would be very helpful, since this remains a significant issue.

There have been some advances in understanding of threshold and lag structures. In the time-series studies, anyway, there is some but only a modest amount of evidence suggesting a significant non-linear relationship, and, particularly, it offers little support for a threshold level for cardiopulmonary mortality at concentrations greater than 20 or 25 $\mu g/m^3$.

There are many studies that demonstrate maximum PM effects after lags of zero to 2 days from exposure. In some studies where longer-term exposures have been studied...evaluated, there is a second peak suggestive of another effect occurring after about 4 days post exposure, and we may be looking at different health endpoints associated

with different lags.

There are also some additional recent studies suggesting effects distributed over weeks or months after exposure, suggesting greater overall excess risk than reflected by the peak lag effects. So, the effect size estimates in the time-series studies may, at least in some cases, turn out to be underestimates of the total effect.

There is a considerable amount of new evidence on cardiovascular effects, much of it associated with endpoints which at least provide insights as to mechanisms and pathways from air pollution exposure to physiological changes. These include effects on cardiac rhythm, effects on blood characteristics, heartbeat, heartbeat variability in panels of elderly subjects, and blood measurements such as increased blood viscosity and serum c-reactive proteins which are both related to increased risk of serious cardiac events.

While this information doesn't completely close the loop between the toxicology and the epidemiology, it certainly points at some interesting directions.

However, there is, at the present time, very little information about which PM components might be specifically associated with a specific cardiovascular endpoint. So, that is a subject for future research.

In the handout, there is a list of examples of new cardiovascular disease studies available for the next draft of the PM document. These have already been published. They will be reviewed and included, and there are a number of other that we are aware of and will include as time permits, and I don't want to take the time to read them all off right now.

There is also some additional information on respiratory effects associated with acute PM exposures. The continuing studies on hospital admissions for COPD pretty much confirms the findings in the PM document and, in some ways, extends those findings.

There are also a number of new asthma studies that show ambient PM exposure associations with increased asthma hospital admissions and visits, and there is new information or newly available information on

non-hospital physician visits. These often have large relative risk estimates, and this suggests that there is a potentially much greater health impact than based on the hospital and clinic records that were used in the '96 Criteria Document.

There is, at least qualitatively, some confirmation of the consistency of the magnitude across various studies. The range of studies in the NMMAPS investigations pretty well cover those observed in other U.S. and Canadian cities, particularly in ranking cardiovascular mortality as having a higher relative risk than total mortality and other studies confirming that respiratory mortality has a higher relative risk than cardiovascular or total mortality, although, because respiratory mortality is a much smaller fraction of total mortality, the amount of uncertainty associated with the respiratory mortality risk estimates is higher than with the other endpoints.

Highest mortality rates in NMMAPS are typically on lag day one. In a couple regions, lag day two or zero appear to have

higher risk estimates overall. Again, whether this is suggestive of other endpoints or not is an open question.

Daily hospital admissions tend to show, overall, a similar pattern with higher admissions for cardiovascular and respiratory causes and overall higher values than the excess mortality risks.

Statistically significant positive associations of 24-hour PM_{10} with daily mortality occurred in the 20 largest U.S. cities in the 90-city NMMAPS Study with various combinations of co-pollutants...this has been published, and we will look at the figures shortly...with the cities taken as a whole. Excess risk estimates in multi-pollutant models are also in the handout just to illustrate the kinds of differences and typical patterns of behavior that are found when the excess risk estimates for cities are aggregated.

There is, however, some tendency for higher relative risks in certain regions, particularly the northeastern U.S. and the industrial Midwest. The reasons for the lower and generally more uncertain effect sizes in

certain other regions, particularly the Southeast and Southwest, are not clear. They may be attributable to study size, that is, the power of the study to detect an effect, and they may be attributable to the greater use of air conditioning in these warmer regions.

Figure 6-10 is...okay, I guess this is for the 90 cities. We originally had this made up based on the New England Journal publication which showed 20-city results. This shows the largest estimate...these are posterior distributions for the mean PM_{10} excess risk based on the NMMAPS results. The distributions take into account the differences between cities and among regions and the internal uncertainty in the risk estimate for each individual study in each individual city.

The highest effects are for PM_{10} alone and for PM_{10} + O_3 which is the dashed line sticking out above. There is a somewhat smaller effect for PM_{10} along with O_3 and SO_2 and considerably reduced effects with O_3 and NO_2 or CO as co-pollutants which is a fairly common finding in previous studies in other

cities.

Now, much of what we have talked about so far has concerned findings for PM_{10}. The specific information for fine and coarse particles has also grown very considerably.

Here, Figure 6-4 from the draft CD shows the those studies where both excess risk for fine particles and excess risk for coarse particles were available. This shows a comparison of the excess risk for the fine and coarse particles for an individual study.

In most cases, the excess risk for the fine particles is statistically significant or, at least, more often than for coarse particles. There are some statistically significant coarse particle effects, for example, for a larger effect found by Lipfert than for the coarse fraction by Lipfert in Philadelphia, by Lippmann in Detroit, a smaller but significant coarse fraction effect found by Mar et al in Phoenix, Arizona, and larger and significant effects for coarse fraction in Mexico City and in Santiago, Chile.

So, there are, apparently, some circumstances in which the coarse fraction,

although not necessarily the crustal particles, but the coarse fraction containing, perhaps, bioaerosols or something else may be accounting for at least some of these effects. Again, based on some of the speciated particle work, it seems less likely that the crustal fraction is contributing to these adverse effects.

The draft document also contains a number of similar figures which I will just bring to your attention. This shows a cardiovascular hospitalization across a number of U.S. studies, predominantly positive effects, many of them statistically significant.

Figure 6-7 shows the risk for respiratory hospital admissions or visits, many of them positive. Let's see. I think, actually, all of them positive and most of them statistically significant for a variety of respiratory endpoints.

Okay, this pretty well wraps up the discussion of the time-series studies. I will not take the time to discuss some recent kinds of studies which I think may prove to be important in the long run, because they provide insight into intermediate time scales,

including, for example, case crossover studies.

Much of the discussion in the document is built around the long-term cohort mortality studies which were also extensively discussed in the 1996 document, and, under the Health Effects Institute sponsorship, these were reanalyzed by Krewski et al at Ottawa; the Harvard Six City study, originally published by Dockery et al in 1993; and the American Cancer Society study, Pope et al in 1995.

The studies included, first of all, an extremely detailed and comprehensive data audit which I should have mentioned but didn't, so that the data quality has, with a few small changes which had little, if any, effect on the end results, confirmed the validity of the data. When the analyses were repeated, the results essentially confirmed the originally published results.

More importantly, there were very extensive sensitivity analyses done for a large number of variables. Substantial changes in effect size estimates of fine particles or sulfates were found on second-stage regressions, primarily for two variables, sulfur dioxide and

education level.

There was a stronger relationship between mortality and fine particles, sulfates, or sulfur dioxide in certain regions, particular the Midwest, Ohio River Valley, and the Northeast.

Some of the figures in the HEI report are extremely informative. I think some people may think that the pictures don't actually prove the case, that only numbers do, but, I think, in terms of suggesting important hypotheses for future investigation, they are very useful.

There was considerable investigation of spatial models in the HEI, the Krewski reanalyses, and there was some sensitivity to modeling methodology. Positive effects were still found, but the magnitude and significance did vary, depending on which methods were used, although there is a problem here in that the spatial aggregation across sites may be at least partially confounding with the fact that some of the ecological variables used in the second-order analyses also change geographically from one region to another across the country.

So, whether or not there is over-adjustment for some of these spatial variables is an open question.

Ecological covariates in the sensitivity analyses which were substantially, again, affecting excess risk from the last to the most polluted cities were education level and average SO_2. The SO_2 levels greatly reduced the estimated fine particle or sulfate effects on total and cardiopulmonary risk. SO_2 may be acting as a surrogate for secondary sulfates which, for many years, have been a major component of fine particles in eastern North America and, in fact, may also be prominent in some other regions.

Excess risk and the statistically most significant for those...the excess risk was most significant and largest for those individuals with less than a high school education, lower and usually significant for individuals with a high school education, and generally not significant for individuals with more than a high school education. It is possible that educational achievement is a surrogate for some other socio-demographic

factor affecting mortality.

Another important new finding was that the relative risk assigned to fine particles or sulfates was substantially reduced but remained statistically significant in the Harvard Six City study which had measured concentrations of sulfate and fine particles throughout the multi-year duration of the study when changes in concentrations were taken into account. This suggests that it may be valuable to consider long-term exposure history in evaluating/assessing prospective cohort studies, since the relative risks were sensitive to the model that was used to take time-dependent exposures into account.

There was also preliminary assessment of non-linearity which, again, provided little evidence against the use of a linear concentration relationship for excess risk, but this, also, clearly requires further investigation.

There were, nevertheless, some spatial relationships that were worth mentioning. Statistical tests showed a significant heterogeneity or spatial variation in the long-

term cohort study, the ACS study, among the U.S. regions. There seems to be some overlap in the regions which turn out to be hot spots, both in the prospective cohort studies, Krewski et al ACS reanalyses, and in the NMMAPS studies, and they tended to cover comparable areas in the industrial Midwest and northeastern U.S., but, again, different methods for spatial averaging in the Krewski et al study did produce different results.

There are a number of other recent prospective cohort studies which I won't discuss in detail, the Adventist Health Study in southern California, the recently-published Veterans Administration study by Lipfert et al which looked at a relatively very large cohort of former U.S. servicemen who were receiving medical care at VA hospitals, and this was certainly a cohort which is worth following up. The Peters et al study of children in 12 southern California communities is also in progress and is starting to produce interesting results.

Finally, I will mention, at least, just...since I have got a minute left here, I

will briefly point out that here is an example of the flood of new research coming down the pike. These were papers that we were aware of before this last draft went out the door. However, when the Criteria Document references were being finalized, they had not been published, and trying to keep to our ground rules of using published research, we were not able to use them. However, these are all drawn in Envirometrics, Volume 11, 2000 November-December issue and indicate a great deal of new work occurring both in methodology and in some substantive findings, both for mortality and for hospital admissions. So, I just wanted to point out that there is a great deal of new research that we are aware of, and we will incorporate as much of this as possible that becomes available in the next few days.

Thank you.

DR. GRANT: If I could just note...direct your attention back to page 15 in this handout, Allan sort of skipped over making note of some examples of new cardiovascular disease studies that are available for the next

draft PM document. These include studies that address an important issue, and that is whether short-term, that is, less than 24-hour, PM exposure effects occur.

You may recall that both Bob Michaels and Mike Klineman, for example, during the last number of years here have been sort of raising that as an issue, sort of a flag, whether or not short-term, maybe a few hour exposures to relatively higher concentrations than the 24-hour average of different PM components might be associated with or account for some of the effects seen in terms of the short-term, so called 24...short-term or whatever 24-hour average studies.

New studies by Peters et al, for example, showing a relationship between the triggering of myocardial infarctions and exposure to PM only a few hours before certainly help direct our attention further at looking at the types of more acute exposure effects. So...and there are a few other studies coming along as well that we are aware of.

I just wanted to signal our intent to,

indeed, try to take on some of those studies. It is sort of a small and limited but, still, now starting to grow kind of database that we need to look at in our next draft.

Anyway, if there are questions or points of clarification that you would like to have regarding the Chapter 6 material, I am sure both Allan and I would be glad to help answer them.

DR. HOPKE: Any clarifying questions?

(No response.)

DR. HOPKE: Let me take a quick break and let Ron introduce himself.

MR. WHITE: Ron White. I serve as a volunteer for the American Lung Association and am currently with the National Osteoporosis Foundation.

DR. HOPKE: Thank you. Okay. Then, I guess, we go back to you.

DR. GRANT: Okay. Allan, would you turn off the 21st century or whatever jet plane, rocket ship?

DR. HOPKE: It shows Allan's extensive stint in Seattle.

DR. GRANT: Right. Now, one of the reasons we are going back to...maybe this is the Stone Age or whatever, but I have been informed that if we run that other machine too long, that rocket ship flames out. Okay? So, Allan, if you could, just please turn it off completely.

DR. MCCLELLAN: Government contract low bid.

DR. GRANT: Something like that. Anyway, back to our old style overhead presentation mode.

Chapter 7 deals with PM dosimetry. We have listed here, just very briefly, a few things that are addressed in the chapter. I am not going to go over all of them. There is discussion about the different human respiratory tract regions.

If somebody...oh, well, why not? What the heck. A lovely castle is back on. Here we go in Windows. We get stuck through screen savers and everything.

In any case, it is very important to note that PM health effects are not just dependent on the external ambient PM

concentrations. Rather, it is very important that they do depend on the actual dose delivered to the target sites. These are determinant by region of specific particle deposition, particle clearance mechanisms, and particle retention patterns and times for the retention in the respiratory tract and/or the transfer of materials elsewhere through systemic circulation or other means.

The deposition, clearance, and retention all depend on particle size, the numbers, and composition of the particles. Total deposition figures are, basically, illustrated on the next slide. I am not going to go over all the information. Those are the respiratory tract regions.

Go to the next one, Dennis. We do have a plot of the total deposition, and I guess the key point there is that below about 0.3 to 0.5, something like that, you get an increase in terms of particle size, increase in total deposition and then also above the 0.3 to about 0.5 micron size, you get an increase in total deposition.

Next slide. It is very important to

note that the deposition patterns are not uniform in each of the different areas, the extrathoracic or tracheobronchial or alveolar regions or within those regions. You do get enhanced deposition in nasal passages, in the trachea, and at tracheobronchial or alveolar branching points or bifurcations in the bronchi or smaller conducting airways.

We discuss in the chapter either actual experimental data and/or modeling data which tend to point towards several factors being of importance in affecting respiratory tract deposition and the regional deposition patterns. We have listed some of them up here, age, indicating probably children being a bit more at risk in the sense both in terms of higher exercise activities and ventilation rates that tend to increase their deposition compared to most adults; gender, some evidence for somewhat greater deposition, perhaps, for females at certain points due to slightly higher normal ventilation rates; disease conditions, a very important factor that we highlight there, that the total lung deposition is increased by obstructed airways, as you find in COPD or

asthma conditions and so forth.

There may also be hot spot deposition at bifurcations even in healthy lung tissue, and that is that we think there is probably a greater particle dose delivered to remaining healthy areas of lung in diseased lungs due to the reduced airflow into obstructed lung areas. So, you may find an increasing impact of particles as part of some of the disease conditions where there is obstruction to one area of the lung or another, the sort of greater deposition impact in remaining healthy areas.

The next slide, please. As far as particle retention, I think there are a few interesting new things. The tracheobronchial region clearance, it has long been known it has both fast and slow components. It is now thought that the slower components may be much more extended than previously thought, perhaps up to about a month, and this enhances the possibility of a more extended period of expected acute exposure health effects. These may help account for the more extended or whatever duration or lag findings or whatever

that have been mentioned in some of the epi work.

The alveolar region clearance involves a number of different mechanisms. One of the interesting things there is that soluble particles deposited in the alveolar region can be very rapidly absorbed into the blood stream and transported to other organs such as the heart so that it looks to be reasonably possible, then, in terms of the clearance mechanisms and removal to other organs, that it would be plausible to have very short-term health effects. As we just mentioned a little while ago, it could be even within a few hours after exposure, as shown by some of the new epi studies.

Lastly, some of the uningested particles deposited in the alveolar region can penetrate into the interstitium and reach lymph nodes within a few hours after deposition. The slow elimination from lymph nodes...some of these now are estimated at half-times of decades, tens of years...along with the focal hot-spot deposited materials, may also provide a long-term internal reservoir of PM-derived

materials from long past exposures that may exert effects long into later life, adulthood and elderly years.

Next slide. This slide just lists all the different areas that were covered, then, in Chapter 8. I am not going to go through all of these. They are there for you to have a look at.

I think one of the very key things is noting that we have new information in terms of both in vivo and in vitro types of exposures and looking at respiratory and cardiovascular effects as key areas; mechanisms of PM toxicity and pathphysiology, also new information on that; and a bit of new information on experimental studies of PM and gaseous pollutant mixtures.

In terms of some of the new things coming out...may I have the next slide there, Dennis...I think lots of folks are interested in where we are in terms of trying to identify potential mechanisms and also any of the bad actors, if you will, in terms of size or composition of PM. I think, as I have put it in quite a number of other public talks over

the course of the last few months, there really...as yet, we do not have clearly identified, shall we call them, smoking guns or a smoking gun that says this particular type of particle, by size or composition, or specific mechanisms are very definitively pinned down yet. That is not the case.

On the other hand, we can also say that it is really quite interesting that we now have quite a number of reasonable hypotheses and more extensive, though limited, new findings on the PM mechanisms and so on.

This is in contrast to the previous Criteria Document back in 1996 in which we, basically, had to say that we really didn't have hardly any even hypotheses, a few perhaps, and very little experimental evidence that you could really call out as supporting the plausibility of the PM epidemiology findings. Now, some of the greater new evidence, new hypotheses and evidence...I just highlighted three different things here from among ones talked about in the document as being promising examples.

Lots of new information on lung injury

and inflammation. Some considerable amount of that information does come from lung instillation of ROFA, that is, residual oil fly ash...it is a fossil fuel combustion product...that does cause lung inflammation in the presence of high content of transition metals, soluble and transition metals such as iron, vanadium, and so on.

You can sort of replicate the effect by using the metals alone. There are also some new studies that look at the inhalation of concentrated ambient particles with only small metal content as showing some mild injury and inflammation at CAPS concentrations of about 100 to 1000 $\mu g/m^3$.

Next slide. Cardiovascular system effects, I think it is worth to highlight that there are now, in addition to some of the different epi results, there is some new experimental evidence which shows some impacts on things such as heart rate variability or blood viscosity or other blood characteristics or particles either administration through instillation or through the concentrated ambient particle type of administration.

Next slide. I think, really, what would be very useful to hear from the committee would be advice and comments on just how best to interpret some of these studies, and I would highlight, in particular, how best to interpret the intratracheal instillation studies. We think they are useful for identifying likely PM mechanisms of action.

On the other hand, there are differences in the patterns of respiratory tract regional deposition and retention from the instilled bolus dose or whatever for the instillation approach versus what you see with inhalation exposures. So, this really complicates trying to extrapolate to any potential ambient exposure-response equivalents.

We also would like to have some comments or inputs regarding how best to interpret concentrated ambient particle or CAP studies. Again, extraction of particles from the ambient air and then reconcentration for exposure, some folks say, may alter the toxicity compared to the real-world mixtures. You are also taking them out of context from being there, perhaps, with the other gaseous

pollutants.

The demonstration of the CAP effects on mortality and morbidity at near ambient concentrations, we think, do tend to enhance the plausibility of analogous epidemiologically-observed effects, but, again, it is very difficult to extrapolate to any likely equivalent ambient exposure-response relationships.

Lastly, we would note that we would appreciate comment on how best to interpret results from some of these new compromised animal models that attempt to mimic human disease states or other susceptibility factors.

The very last slide as far as Chapter 8 is just to list a couple of examples of some new studies becoming available now on the toxicology side that we certainly intend to include.

Very importantly here, quite interesting from our own EPA laboratory's Dan Costa and other associates, Andy Ghio and so on, Bob Devlin as well as other authors listed there.

These are some studies in which the particles collected from the Utah Valley both

during the operation of a steel mill there and then during a period when it was shut down and then, again, afterwards when it was restarted that those particles, filter extracts, were then taken and used to expose rats, I believe, through instillation approach or whatever, and finding, then, increased inflammatory lung injury from the particle extracts taken during the operation of the steel mill but then not while the steel mill was closed down but then again after it reopened, again, those particle extracts showing inflammatory responses, if you will, in essence, a natural experiment or whatever.

We think that tends to add some substantial plausibility to the epidemiology findings regarding, for example, from Popes' epidemiology studies and probably other ones, that, indeed, the ambient particles, including metals, combustion products or whatever, are likely involved here in producing untoward effects.

The very last slide is one that deals with Chapter 9, the so-called integrative synthesis chapter. First thing for me to note

that it really does not yet represent integrative synthesis. It is now mainly a preliminary summary, if you will, drawn from, you know, these other chapters. To be frank, we simply ran out of time.

Believe me, trying to pull together the massive amount of new information that went into the other detailed chapters and resetting, several times, when we thought we would be able to put that document out, we finally just came to a point and said okay, we'll try to summarize, to some extent, some of the key points out of the other chapters in this one, and let's get it out the door, let's get the discussion by CASAC of the detailed chapters and public comment on them, and then come on back around and really try to put together the integrative synthesis.

So, we are considering that perhaps to use the basic framework that we had in the '96 Criteria Document, that integrative synthesis which the committee found to be quite well done or whatever and perhaps update it to reflect the newly available information in this current Criteria Document. I will just note

that we do intend to include in there discussion of risk factors in susceptible subgroups and likely to include those following items, the age, the gender, preexisting disease conditions as highlighting types of risk factors or whatever.

Lastly, I should note we will be putting together an executive summary. That was lacking in here. It didn't seem to make very much sense to try to pull together a very brief, succinct, hard-hitting executive summary until we would have the benefit of the review of the other more detailed materials in the document and take advantage of the new studies that we are going to consider up through July in the next draft, but we do intend, then, in the third external review draft to have a version of the executive summary.

I would be glad to take questions or answer any need for clarifying points or whatever with regard to these last several chapters.

DR. HOPKE: Any quick clarifying questions? Ron?

MR. WHITE: Les, what is the

date for cutoff of studies for the next draft of the CD? Do you have a date at this time that you anticipate?

DR. GRANT: Yes. We noted that we tried to be as inclusive up through December in this draft, but we still missed some studies coming out late last year. We will be taking those into account that we missed. We will also go ahead all the way through the month of July now, and, basically, anything actually published or accepted for publication by the end of July, that is what we are considering the cutoff point for incorporation into any of the next drafts of the document.

The only proviso there, as mentioned earlier, is if there is some truly monumental, truly significant incremental new set of findings that might have a big impact on some bottom-line conclusions and so on, then we would have to consider and probably consult with the committee as to whether to bring those into play as well.

DR. HOPKE: Okay. It is now 9:56. We will take a 9-minute break, start

promptly at 5 minutes after 10:00 at which point we will start the public comments. The first public speaker is Fred Lipfert, and, Fred, if you could be ready to go at that time, I would appreciate it.

(**WHEREUPON**, a brief recess was taken.)

DR. HOPKE: We are going to be very rigorous with the time, and I apologize for cutting people off, but we will give you 5 minutes. We have 30 people to hear from today between now and lunch and the one hour after lunch. Therefore, we have to be very rigorous with the time interval. I will try and give a 30-second warning, and then we will cut you off at 5 minutes.

If there is a really pressing question from the panel that really needs to be answered in order to clarify things, then we will take those questions, but keep in mind that we do have detailed written material from each of these people so that we have an opportunity to get a lot more than can be presented in the 5-minute highlight. So, please, let's try and work to keep things that way.

MR. FLAAK: What I would like to do is ask...I have a lot of the comments from the individual speakers already. For the remainder of you that still have individual comments to pass out, please hold onto those, and when your time comes to speak, please bring those up to me, and I will distribute them.

I will ask that the speakers, as they come up, the next speaker please be ready to come up quickly so we can move along smoothly and get up to lunchtime having gone through the majority of these. The next speaker is Dr. David Chock, also, after Dr. Lipfert.

So, Fred, are you ready?

DR. LIPFERT: Yes, sir.

MR. FLAAK: All right. David, are you ready? Where are you? Okay, great. Thank you.

DR. LIPFERT: Good morning. First of all, let me express my appreciation for this opportunity to speak. This talk is on behalf of the Alliance of Automobile Manufacturers. The Alliance is a trade association of 13 members which represent over

90 percent of U.S. vehicle sales.

What I am going to try to do is to summarize our EPA comments which are quite voluminous.

The first point, first set of points I want to talk about are the conclusions from Chapter 6. There were 15 of them, and we picked up the 5 here that we thought were particularly important. Let me just try to run through them quickly.

With respect to the separate effects of $PM_{2.5}$, we would say that, in looking at decent data for the coarse fraction, that question is going to be open.

That brings us down to here, that both size fractions are associated with hospital admissions. This is from the CD, and if that is the case, and we think it probably is, then you really have to go back and rethink the whole scenario which was built strongly about fine particles in terms of plausibility and in terms of monitoring data. So, if we have both fractions, we really have to run both things parallel. That hasn't been done.

With respect to chemical and physical

properties, we have to say that the toxicology really doesn't mesh very well with the epidemiology. Confounding by co-pollutants is strongly tied up with measurement error, and it is not just a spatial error on the ground. It is the exposure error to the target organ that has not really been considered fully.

Finally, in terms of the heterogeneity, there is large heterogeneity. In NMMAPS, it is several factors the CD cites in order of magnitude in the PM exposure.

So, let's talk about that a little bit more and look at some actual data which I hope you will find informative. These are the eight most...sorry about that...this is the PM_{10} regression coefficient for the eight most significant NMMAPS cities, and I chose those eight so you could...so it would be clear, and they are plotted against the mean PM_{10}.

What it shows is there is a negative relationship. The dirtier the city, the smaller the effect. So, if this were true, if we believed this, it would say that cleaning up is going to be counterproductive.

Now, bear in mind this point. It has

a slope of -.15. That is just eight cities. Let's go ahead and look at the rest of them, and we'll do that in a slightly different way.

Here, we have plotted the slope of the line like the one we just saw. So, the point you just saw is here, 0.15, and to put this on a log scale, of course, we had to make the coefficient negative. So, what I have done is look at what happens to this relationship between the strength of the PM_{10} effect and mean PM_{10} value as you add more and more cities into the mix and their rank in decreasing T values. So, over here, this is negative.

The difference between the standard error and the coefficient on a log plot is the T value. This is about 4 all the way up here to 65 cities. So, for 65 out of the 88 NMMAPS cities, we have a negative relationship between the magnitude of the effect and the magnitude of the pollution.

Now, it has been proposed, for example, by Levy et al that this is due to variability of $PM_{2.5}$. So, we looked at that next, and we did it the following way.

There are, of course, no $PM_{2.5}$ data that

are parallel to the PM_{10} data. If there were, they would have been used in NMMAPS. We took the 1999 ambient data from the league network and ratioed that against PM_{10} using NMMAPS and used this as a candidate explanatory factor for heterogeneity, and as you can see, it doesn't work either for all cities which are the red dots or for the most significant ones which are the blue ones.

So, we would have to conclude from this that there is a lot of heterogeneity. $PM_{2.5}$ is not the answer for this particular data set.

So, what are the implications of all this? This is our take on implications.

DR. HOPKE: 30 seconds.

DR. LIPFERT: Thank you. First of all, think about this. There is nothing in the CD and I have never seen anything published that would tell me why a person who has been exposed over and over again during his entire lifetime to some level of air pollution would suddenly, on some particular day, experience a health effect. Well, the answer is because his health has deteriorated,

but that factor has not really been considered.

The confounding question is with us very much...

DR. HOPKE: Time.

DR. LIPFERT: ...and we don't know what role it plays. I hope you can read that.

DR. HOPKE: Yes, Petros?

DR. KOUTRAKIS: Yes, please, go and to look at the paper in Environmental Health Perspectives where all the NMMAPS coefficients for mortality and morbidity were studied, and most of the variability was explained by 76 percent of emissions from cars, and the other 8 percent from the air conditioning, and all these low numbers you have there can be explained by that. So...

DR. LIPFERT: I'll take a look at that, Petros. Thank you.

MR. FLAAK: The next speaker after Dr. Chock is Dr. Schwartz.

DR. CHOCK: My name is David Chock. I am an environmental research scientist at Ford Motor Company. I want to thank the EPA for the opportunity to share

with you two findings that I believe are significant in the setting of air quality standards.

The two findings are summarized here. First, the draft Criteria Document contends that statistical causes alone lead to a high correlation between the community average PM exposure and the ambient PM concentration. In terms of these statistical process activating non-statistical functions has yet to be confirmed. Therefore, the contention that epidemiological models will not be biased by the non-ambient confounder of PM exposure is premature and remains to be substantiated.

Point number two, measurement errors caused by use of ambient PM concentrations in place of personal PM exposure can not only mask the presence of a true particle effect on threshold but, also, should the apparent true particle correspondence threshold shift the apparent threshold away from the possible true threshold that is based on personal exposure. Therefore, the general contention that there is no PM health response threshold is premature.

The true health response threshold

cannot be determined by epidemiological studies using only ambient concentration data, and we go into these issues in more detail.

The disputed contention is based on the work of Hall et al which assumed that personal PM exposure...which assumed that personal PM exposure is the sum of the non-ambient contribution and the ambient general contribution as in this slide here. The latter, which is this point here, is proportionate to ambient concentration Y_{ij}. Hall et al then assumed that ambient general contribution is independent of individuals. In other words, all houses and buildings have the same filtering efficiency of the ambient air, et cetera, and the ambient concentration is spatially uniform which is given by this point here.

Of course, all these assumptions are to be substantiated. With these assumptions, one can average over individuals, i, and reduce the factors containing individual variation to a constant, resulting in community-averaged PM exposure becomes highly correlated with the ambient-generated contribution.

But is this necessarily true? The answer is no, because averaging one individual...averaging all individuals does not get rid of the day-to-day variation of the non-ambient contribution unless we further assume that the non-ambient contribution does not vary from day to day as described in here.

If we make these assumptions, then we will have to see this assumption will have to be able to conform as well, but, interestingly, with this assumption, one can also average over the day-to-day variation of ambient PM concentration and come to the conclusion that, for a given individual, the time-averaged personal PM exposure is highly correlated with the non-ambient component of PM. This conclusion sounds controversial, but it is a statistical consequence of these assumptions.

The CD contention further necessitates the assumption that the health response is a linear function of PM concentration, but this assumption is not necessarily valid based on the results of some locally-medium models and generalized epi models. To ascertain how measurement errors due to using ambient PM

concentration in place of PM exposure affect the characterization of threshold based on personal PM exposure, we assume that personal exposures and average concentration are described by or are correlated by Garrett log-normal distribution and assume that the threshold of 25 $\mu g/m^3$ is present in the personal exposure.

When the correlation is 1, the threshold in the ambient concentration can be readily detected, but the threshold value shifts, depending on how the concentration mean and standard deviation change relative to those of the personal PM personal exposure which is presented here. There is a plot here of the mean versus standard deviation of the ambient concentration, and the threshold tends to shift away from 25 $\mu g/m^3$, depending on the relations between the mean and the standard deviation relative to the PM exposure.

DR. HOPKE: 30 seconds.

DR. CHOCK: As we lower the correlation coefficient, the threshold becomes difficult to detect as low as 900 geometric standard deviation type and as low as 0.6 to

0.7 when the geometric standard deviation is low. These values are all near the upper range of the studies of slope by Samet et al, and here is an example. And those near the 1 can see the threshold, and those near the...

DR. HOPKE: Time is up.

DR. CHOCK: ...and a coefficient 0.8. Thank you.

DR. HOPKE: Next, Dr. Schwartz.

MR. FLAAK: The next speaker is Dr. Moolgavkar.

DR. HOPKE: After you. On deck speaker.

MR. FLAAK: On deck speaker.

DR. SCHWARTZ: I would like to present the results of three press papers that I think might be relevant to the Criteria Document. One deals with the issue of confounding by gaseous air pollutants. There is considerable discussion of that in the CD, and it is based on the assumption that PM_{10} and gaseous air pollutants, measured ambiently, are surrogates for their personal exposure.

We have a lot of new studies coming out showing that that is true for PM,

relatively little talking about the gases. So, this is a study Garam and Sarnak did in 56 subjects with 12 consecutive days of measurement in the summer and the winter and personal measurements of gases and particles.

What we see is that there is a significant association between ambient PM and personal PM in the summer and the winter overall and personal of ambient origin. So, that surrogacy exists.

However, ambient ozone is not a surrogate for personal exposure to ozone. Ambient SO_2 is, in fact, negatively correlated with personal exposure to SO_2, and personal ambient ozone is not a surrogate for personal ozone.

So, what are daily variations in these ambient gases surrogates for? Well, they are surrogates for particles. The ozone is associated with personal PM, as is the NO_2, the SO_2, and the carbon monoxide, and the association of ozone with exposure to particles is negative in the winter.

First conclusion: inappropriate to treat a variable as a confounder of another

when they are both surrogates for the same thing. So, the whole discussion is, I think, off base. You need to measure exposure to gases to figure out what is going on.

Also, notice that the personal gases are not correlated with the personal particle exposures, so they can't possibly be confounders, because there is no correlation of exposure.

The next thing I would like to talk about is harvesting. The notion is air pollution goes up, people die today, and they would have died a week later. If that is true, there is a negative correlation between deaths a week from now and exposure today. That is a testable hypothesis.

We took ten cities in Europe with a population of 28 million people, and we put in PM_{10} today, yesterday, up to 41 days lag, simultaneously together, unconstrained, 41 variables in the model. We added up the overall effect, we did a meta analysis, and the net is that we go from a baseline estimate similar to the NMMAPS estimate to 2.5 times higher estimate, not less, when we take into

account the lung effect which is something that Lester talked about in terms of deposition.

The last study is looking at dose-response relationships in eight cities in Spain. We picked non-parametric smooth curves for all of them, combined them all together, looked at the dose-response. It looks quite linear, but it has actually got a steeper slope at low levels which would explain the negative correlation between mean concentration and regression coefficient within the cities.

SPEAKER: BS is British smoke?

DR. SCHWARTZ: BS is British smoke. That was the one that they had the measurements on in the most cities, so that is what we did.

And when we control for SO_2 simultaneously as a smooth function, there was no change in the slope. Here is what I showed you before, here is SO_2, and the third curve is when I did the analysis similar to the way the NMMAPS did it and fit the same smoothing parameter for temperature and season in every city instead of fine-tuning this. That turns out not to be an issue as well.

Thank you.

DR. HOPKE: Quick questions?

(No response.)

DR. HOPKE: Okay. The next speaker, then, is Dr. Moolgavkar, and the on deck...

MR. FLAAK: On deck speaker is Mr. Heuss.

DR. MOOLGAVKAR: Okay, I am going to make comments on Chapter 6. It is impossible to do justice to this 300-page chapter in 5 minutes, so I am going to omit all my positive comments.

MR. FLAAK: Thanks, Suresh.

DR. MOOLGAVKAR: This is not really a critical review of the new literature since the last CD in 1996. It is a pretty complete catalog of studies with little critical discussion of each. There is a lot of gerrypicking of results to support the EPA position in the 1996 CD with no attempt at discussing the considerable heterogeneity of results that have been observed since then.

It is a comprehensive catalog of studies. Yet, the final interpretive synthesis

draws heavily on just a few studies with no clear justification of why those studies were chosen for special attention, and more details with specific examples of each one of these blanks are given in my written comments that were sent to EPA and also to each member of CASAC separately.

Now, I would like to say a few words about the NMMAPS studies, because the interpretive synthesis depends heavily on the NMMAPS studies, and I have some problems with some of the technical aspects of the studies and also with the interpretation.

First of all, the method used to control confounding in the morbidity of the hospital admission studies has completely unknown properties, and power of this method is likely to be low as was also remarked by the HEI Review Committee, although not quite as strongly as I am making the comment now. These studies should be considered to be single pollutant studies. They cannot be considered to have adjusted for confounding.

Gases were not given equal treatment with PM in the mortality analyses. I would

like to know what would the posterior distribution for the gases look like if they were considered in the same way as PM. How would that change the interpretation of the PM posterior distributions, and what about biradiant base analysis with PM and gas at the second stage as was done with ozone in the 20 cities analysis?

Ozone, in fact, had the weakest association with mortality, and, yet, it was chosen in the second stage analysis for a base analysis, but the other gases were not. I would like to see what would happen if the other gases were included.

And I am grateful to Jon Samet for having made the data available so that I can look at some of these issues in the near future.

The results depend strongly upon the prior distributions chosen. In fact, a normal prior was chosen, and looking a priori at the results from the individual cities, I would have chosen a mixture of two normals, because about 30 of the coefficients are either negative or close to zero. Why choose a

normal prior? That determines the outcome. Why not choose a mixture of two normals? Much more difficult analyses, but that shouldn't stop us from doing it.

I think that the interpretation of the posterior distribution as PM effects is questionable, and the details of this are quite technical, and they are given in my write-up. First of all, basing analysis on type of priors are often very difficult to interpret.

With respect to exposure-response relationships, one picture is worth a thousand words, as I say here, and I have given another example in my write-up, but here is an example right here. I have got for this write-up, but you saw it.

Here are exposure-response relationships from Cook County. The lags are between zero and 10 for PM_{10}, and I chose Cook County here, because PM_{10} measurements are available on a daily basis, and you can see that these exposure-response relationships are difficult to reconcile with any biologically plausible hypothesis regarding the effect of PM on daily mortality.

So, what are my conclusions? My conclusions are that it is time to address air pollution as a mixture with thousands of components. I think it is naive to interpret regression analyses...

DR. HOPKE: 30 seconds.

DR. MOOLGAVKAR: ...with five monitored components as representing the effects of the single component in the regression, and the conclusion I would come to is that epidemiological studies appearing after 1996 confirm that air pollution indexed by PM and/or gases is associated with diverse health effects on human health even at levels of pollution found in contemporary U.S. and Canadian cities. These studies cannot identify the actual components...

DR. HOPKE: Time is up.

DR. MOOLGAVKAR: ...of the air pollution mix or the fraction to be attributed to them.

DR. HOPKE: Thank you. Next, please.

MR. FLAAK: The on deck speaker is Dr. DeLucia.

DR. HEUSS: My name is Jon Heuss, and I reviewed the CD for General Motors.

The new studies that I reviewed on the CD fail to support the Agency decision to focus on fine particles. There are many new time-series studies that we've seen, but many do not implicate PM as the sole source or even the independent cause of the effects.

There is evidence of significant confounding by other pollutants, weather, as well as evidence of false positives. Most importantly, modal studies in the same city do not produce the same result. In addition, it is a major error to assume that exposure to PM of ambient origin is independent of exposure to PM of indoor origin.

Because of these inconsistencies, the Agency cannot identify ambient PM as the single factor responsible for the reported effects. To the extent particles are involved, both fine and coarse, they are intermingled.

The CD doesn't rigorously evaluate the consistency within the epidemiology, and it doesn't discuss consistency with PM risk in

other settings at all. It doesn't acknowledge the presence of false positives, and, here, the NMMAPS ozone results are probably the best example. Although there was no overall association with ozone, there were many cities with individual positive associations.

Season is another major issue. Several investigators have demonstrated the importance of seasonal effects, and season is important because of the correlation among pollutants and between pollutants, and weather is season dependent. There are also seasonal differences in pollution levels, PM composition, air exchange, and human behavior. NMMAPS should analyze all pollutants by season as they did for ozone.

There are now five studies of mortality in the last decade in Los Angeles and four hospital admission studies. In 1991, associations were reported with a number of pollutants, but a measure of fine particles was not associated. In 1995, positive associations were reported for ozone, PM_{10}, and CO with mortality, and in models with PM_{10} and ozone, the ozone effect went to zero. In models with

PM_{10} and CO, both coefficients were positive. In 1995, there was another study that estimated $PM_{2.5}$ that showed association only in the summer.

NMMAPS in the 20 cities also studied Los Angeles. There was an association with PM_{10} with ozone...with PM_{10} but not with ozone, and in the three Philly models, the PM_{10} coefficient went to zero. Moolgavkar, interestingly, had a study in Los Angeles on mortality that came, essentially, to the same conclusion.

All five of these studies reported some association with mortality. However, they don't agree on the air pollutants involved; they don't agree on health endpoint affected. When you look at the four hospital admission studies, they also do not agree.

These inconsistencies that happen both in Los Angeles and other locations where the mode of studies are a severe impediment to use of the data to make any policy decisions.

The CD dismisses indoor pollutants by arguing that exposure to PM of ambient origin is independent of exposure to PM of indoor

origin. They argue that daily activities are independent of weather. They use analyses assuming independence, and they use some PTEAM herbicide data.

But daily activities and emissions that lead to both indoor and outdoor PM are independent of weather. Daily changes in weather drive outdoor pollutant concentrations, but they also influence air exchange rates that determine the exposure to indoor pollutant sources. The PTEAM results are from a cross-sectional study in an area with very high air exchange rates. These factors mask the association from a longitudinal study in an area with typical air exchange rates.

In naturally ventilating buildings, weather affects air exchange based on wind and temperature-driven pressure differences.

DR. HOPKE: 30 seconds.

DR. HEUSS: So, reductions in wind speed will increase ambient PM exposures, reduce air exchange, and also increase indoor pollutant exposure. This degree of confounding can be evaluated by EPA's indoor and outdoor models using standard ventilation information.

We have an example in our write-up.

MR. FLAAK: Debbie, you are on deck.

DR. DELUCIA: Good morning. I am Dr. Anthony DeLucia and I serve as President-elect of the American Lung Association. I'm here on behalf of a number of health organizations, including the American Public Health Association, the American Academy of Pediatrics, and the Asthma and Allergy Foundation of America, and several dozen public interest environmental organizations. Collectively, we represent millions of Americans who are concerned about public heath risks of breathing particulate matter air pollution, commonly known as soot, or haze. We believe that strong air quality standards are the first step to alleviate the suffering of children, the elderly, and people with heart conditions and respiratory diseases such as asthma who are most susceptible to the effects of fine particle air pollution. We believe that the public health payoff of strong air quality standards for fine particles will be enormous. While new research is advancing our

understanding of particulate air pollution every day, we believe the current science necessitates moving forward without further delay to protect the health and lives of our most vulnerable citizens. We strongly support EPA's approach of focusing on new developments in the scientific literature since the last Criteria Document was published in 1996. This research leaves no room to weaken the air quality standards adopted in 1997 and, indeed, makes a strong case that the short-term fine particle standard needs to be strengthened. We also believe the underlying health evidence dictates the establishment of a meaningful coarse particle standard.

Hundreds of scientific studies have been published in the last five years as a result of research programs which have been carefully coordinated in order to advance our understanding of the most important scientific issues and to address the primary arguments raised by industry critics. Taken together, the studies confirm the relationship between particle air pollution, illness, hospitalizations, and premature death and

emphasize the following points. The major long-term mortality studies have been intensely scrutinized and fully validated. Six dozen new short-term studies from across the United States and around the world confirm the effects of particle pollution on premature mortality, hospital admissions, emergency department visits, doctor's visits, respiratory and cardiac effects. Recent laboratory and chamber studies of animals and humans, as well as epidemiologic studies of cardiac effects, have elucidated possible biologic mechanisms. I have already commented with regard to vulnerable populations.

Careful examination of factors such as weather, other air pollutants, socioeconomic indicators and other environmental variables have eliminated them as factors accounting for the relationship between particle pollution and mortality and morbidity. For the most part, we believe that the Criteria Document and Staff Paper do a good job of summarizing the research advances of the last several years. In addition, significant progress has been made in addressing some of the scientific uncertainties and the allegations made

concerning the 1997 standards. For instance new scientific research has refuted contentions about mortality displacement or harvesting. Findings from short-term studies may indicate life shortening of weeks or months and long-term studies may show life expectancy curtailed by a year or more. The NMMAPS study has shown that the exposure measurement error would likely cause an underestimate rather than an overestimate of mortality risks associated with PM10 exposures.

Most of the new studies have examined other common air pollutants and found that the association with particulate matter remains strong. Independent re-analyses of the long-term studies have exhaustively considered potential confounding variables and alternative statistical models and have concluded that the association between fine particles and mortality are robust. Importantly, new advances on the source attribution of particles have identified combustion source particles from power plants and motor vehicles as those most closely associated with death and disease. Again, we believe that the Criteria Document and the

Staff Paper do a good job of interpreting this literature.

Finally, we would like to briefly offer our perspective on some policy issues to be addressed in the next draft of the Staff Paper. First, the extensive re-analyses of the long-term studies has confirmed that the annual average standard for PM2.5 established by EPA in 1997 was set appropriately. This standard must not be weakened in any way. Second, in 1997, EPA set the 24-hour PM2.5 standard at the upper end of the range at 65 micrograms per cubic meter. This standard is so weak that it will require only a handful of areas to reduce daily concentrations, even though hundreds of studies have now established a relationship between lower particle levels, death and disease. In 1999, when EPA established a public warning level for fine particles, it set the Air Quality Index at 40.5 micrograms per cubic meter, acknowledging the fact that 65 inadequately protected susceptible members of the population. The existence of a public warning level in no way mitigates the need for a stronger 24-hour

standard. We will be giving close scrutiny to the ranges suggested for the 24-hour standard in the next version of the Staff Paper. Third, we understand that EPA will likely establish a new coarse particle standard to replace the PM10 standard as directed by the Court of Appeals. While studies reporting effects of PM10 minus 2.5 do exist, we believe it's important that the massive number of studies documenting the effects of PM10 also be considered in establishing the new coarse particle standard. In our view this approach best offers the public adequate protection. Thank you for the opportunity to put forth our views.

DR. SHPRENTZ: Good morning. I'm Dr. Shprentz and I serve as a consultant to the American Lung Association. The review of the NAAQS for PM is one of the most important environmental health decisions facing EPA. Each year, an estimated 50,000 people die prematurely due to particulate air pollution and there are tens of thousands of hospital admissions, emergency room visits, and cases of respiratory problems. The elderly,

infants and children, people with pre-existing heart and lung disease are those at greatest risk. These are the people that the American Lung Association represents. We would like to commend the EPA staff for its thorough and comprehensive review of the recent scientific literature. In brief, the Lung Association believes the new scientific information supports reaffirming the annual average fine particle standard, strengthening the 24-hour fine particle standard, and setting meaningful new standards for coarse particles.

Today I want to focus on the 24-hour PM standard because we believe that's the area most in need of review. Both the level and the formula standard set by EPA in 1997 was not sufficient to protect the public health, particularly the health of the sensitive population. I would like to discuss the 24-hour standard in terms of the three key issues that are before CASAC today. 1) the need to update and broaden the analysis of the new PM fine monitoring data; 2) the need to broaden the proposed risk analysis; and 3) the need for more interpretation of key studies in the

CD and Staff Paper.

First, let me talk about the monitoring analysis. When EPA established fine particle standards in 1997, they assumed that the annual average standards would be sufficient to protect against peak short-term concentrations and as a result EPA set a very lax 24-hour standard. At the upper end of the staff record is the grading and a very relaxed form of that standard, a 98th percentile form, which leaves the public unprotected from air pollution on the most polluted days. ALA did an analysis of the 1999 fine particle data which did prove the EPA's assumption. You can see the results here. This is only from monitoring stations where the data was 75 percent complete or greater. And we looked at cities that have annual average concentrations under 15 units that had high 24 hour concentrations. You can see that there are a number of major cities with concentrations, 24-hour concentrations above 40.5, EPA public warning level for fine particulates where millions of people live but they would be unprotected by EPA for more than 24 hours.

Over 60 additional cities where 24-hour concentrations are above 25 micrograms, but, again, are unprotected by either the 24 or the annual average standard. We'd like to ask members of CASAC to request that EPA take a look at this testimony and analyze the 2000 monitoring data.

In addition, a couple of other key questions. How high do concentrations go? EPA shouldn't just be looking at the 98th percentile. How many days are these areas experiencing high concentrations? Key additional areas we'd like to see included in the monitoring analysis. Second, with respect to EPA's proposed risk analysis, we think there's a clear need to broaden the geographic scope of the analysis. At a minimum, to look at the major American cities that have been the subject of extensive research on particulate matter precisely because they have good monitoring data. We believe there is also a need to look at other facts such as chronic bronchitis, infant mortality, and in addition to analyzing the public health impact alternative levels of the standard, the risk

analysis should also look at alternative, the public health impact alternative forms of the standard. For example, allowing one exceedance versus multiple exceedances. And, finally, the whole purpose of this exercise that we're engaged in here is to review the adequacy of the current air quality standards to protect the public health including sensitive populations and we believe in order to accomplish this objective, the key studies need to be discussed in terms of the '97 standards. Are the new studies finding effects at levels below the current standards? Are they finding effects at contemporary concentrations experienced in the United States? These are the key questions that need to be explicitly addressed in the summary tables in Chapter 6 and Chapter 9. Thank you.

DR. HOPKE: Next. Previously, Fred was representing the Alliance of Automobile Manufacturers. In this particular presentation, he's representing himself. The next speaker up is Dr. Michael Halpern.

DR. LIPFERT: Thank you and good morning again. Please don't blame anybody

else for these remarks. They're coming from me. I'm here today on this talk essentially as an aggrieved author. I want to complain about the shabby treatment that I think four papers received in the CD and explain to you why they need to be revisited. This one while I was not an author, I was heavily involved. It has to do with harvesting. It involves an entirely new methodology for dealing with this issue and was not mentioned anywhere in the document. It was presented in Charleston, sent to EPA, and it's hard for me to understand why it was ignored perhaps because the answer is the harvesting effect is two and a half days.

The next one was published and cited in Chapter 6, but not in Chapter 9, it has to do with infant mortality. My main point here is that while we found this... This was prompted by the work of Woodruff, et al., which emphasized Sudden Infant Death Syndrome. We found a similar result with Woodruff when we used her methods and data, but we found a lot of other things. The most important thing we found is that sulfate has an enormously large negative effect. Now, we don't believe

that. And you don't either. And if you don't believe that, you can't believe this. The reason for this is because neo-natal mortality is much higher in the western U.S. than it is in the east. It's been that way for decades. The idea of telling a parent of a SIDS casualty that that death might have been due to PM10 I think is irresponsible. Now, if you do a risk assessment on infant mortality figures, then you either have to use all the pollutants or none.

This is a tale of two cohort studies and you may recognize some of them. I just want to point out here that there's a big difference between this study, which got a big play in the Criteria Document, and this one which got essentially no play. The differences are the number of locations, the type of data which is epidemiological data. The main thing is that we used past, present and future exposures. We had age interactions, if not, we had a non-linear model. Now, if you just look at this. Which study would you choose? I won't wait for your answer. I'll just explain to you why it happened the way it

happened. And I have a shot here. Of course, that was the six city study, this is the VA study. This study finds a large significant positive effect of PM.2.5 and sulfate. This finds a large significant negative effect of those two pollutants. What we did find, that wasn't discussed, is the significant effect of peak ozone which was not evaluated in either ACS or Six Cities with the threshold, and I urge you to read the study, it's in Inhalation Toxicology.

Finally, the fourth one had to deal with time series in Philadelphia, where we looked at 75 different PM metrics, and a bunch of other things. Our main finding in this paper was that ozone was the most important pollutant in Philadelphia and this combination of ozone with fine and ozone with coarse were different than either one together. This number was in Chapter 9. I have no idea where it came from. We found no statistical significant sulfate results for that traffic area, nothing even close. So, again, this study was taken out of context, important findings were not cited and that was this

data.

So since I seem to have a few seconds left, let me just say that what's at stake here is the credibility of the process. Industry is putting millions of dollars into this research with the good faith understanding that it would get the same treatment as everybody else. Clearly this is not happening and I would say that you, CASAC, I would urge you to urge EPA to understand the difference between doing the science, which Congress wants us to keep, and defending a regulation, which is what the CD does. Those are two different activities and it's important. It's just not my complaints here. The way I see it, the credibility of regulation in this Country is at stake if we can't resolve this.

DR. HOPKE: Okay, our next speaker on deck here is Dr. Resha Putzrath.

DR. HALPERN: Good morning. I'm Dr. Michael Halpern. There have been a number of recent studies on the reanalysis of immunology PM health effects. Generally, I believe these have been well done, objective studies that have provided pertinent

information. However, premature conclusions in a CD often distort the results of these studies, provide incomplete results and provide an unbalanced summary of the overall studies. This lack of balance in the CD is most visible in two areas. First, the role of potential confounders, including co-pollutants on the association between PM and mortality. The second on model discussion, especially models that are used to evaluate the association between PM and mortality.

There are a number of steps that the CD needs to take in order to achieve balance. First, the CD must report the complete information and findings, to include negative findings regarding the meaning of copollutants, with regard to a series of other possible unspecified factors and approaches and subjects used in model selection. Second, CD must include results from all assessed risk factors in an objective, unbiased manner and consider these results and include negative results to make its conclusions and recommendations.

Let me cite some of the recent studies that provide some support for my conclusions.

Clearly, PM is just one of numerous co-pollutants present in the atmosphere, however, the CD tends to minimize the importance of these co-pollutants. I quote here a statement in Section 6-2 which states that PM/mortality associations are not seriously distorted by co-pollutants. That clearly is not the case if you look at some of the recent literature. In the ACS reanalysis, inclusion of SO2 to extend a base model reduces the magnitude of the relative risk for PM associated mortality and makes the relative risk estimate there non-significant. Clearly, in the MF 20 City study, additional co-pollutants reduced the number of cities that had positive and statistically significant regression coefficients from seven to zero in some free pollutant models. Simultaneously, the number of cities with negative albeit non-statistically significant regression coefficients increased with the increase in co-pollutants.

Other variables besides co-pollutants also have substantial impacts on these apparent associations between PM and mortality. In the Harvard Six Cities reanalysis, gender and

education level had very substantial impacts here. Education level is perhaps the most important. If you look at a population that has more than a high school education, the relative risk becomes non-significant and is actually less than one. Based on those results, either these variables, in this case in the Six City reanalysis, gender and education level, directly influence mortality associated with PM exposure. I don't believe that to be the case. Rather, I believe that these variables are correlated with other unmeasured and unspecified variables such as health risk behaviors, health related activities that make moderate changes in mortality risk. Therefore, the models currently evaluating association between PM and mortality are incomplete, are missing terms and these unspecified variables once included in the models may further decrease the apparent association between PM and mortality.

Choice of an appropriate model to evaluate the association between PM and mortality is also clearly an important issue. An appropriate, objective, unbiased model is

needed. This was clearly stated at one point in the CD, that the fundamental issue is the selection of an appropriate statistical model in the absence of any strong prior hypotheses. Unfortunately, the CD doesn't always follow its advice. On the very next page, on page 6-218, the CD states that in general one would expect the best PM model to begin would be models with the largest and most significant indices. A priori selection of the model based on desired characteristics, in this case, a large and/or statistically significant PM regression coefficient is going to lead to biased results and specious findings.

In conclusion, I believe there's still multiple areas of concern to you in making a reported association between PM and mortality. To address this concern, the CD must focus on the highest qualify objective approaches, model selection for inclusion of co-pollutants and for evaluation of potential unspecified factors. Further, the CD must make conclusions and recommendations based on comprehensive findings from all assessment factors and potential confounders in an unbiased manner including

studies with negative results. Thank you.

DR. HOPKE: The next speaker up after Dr. Putzrath is Dr. Harriet Ammann.

DR. PUTZRATH: I'm Resha Putzrath, Georgetown Risk Group, and I'll be addressing Chapter 8, the chapter on toxicology. I had some problems reviewing this chapter starting with the overreaching problem of trying to decide decision rules and criteria for making selections and condensing the data that are available to the chapter. For example, one of the major purposes of the toxicology chapter is stated to be to address what are the possible biological plausibilities or causalities for the effects of interest. But how do we determine the effects of interest? One suggestion, of course, would be those that are found in epidemiology studies. But if this is the case, then I have a hard time understanding the great emphasis put on the immunological effects as these are, at best, a minor and inconsistent finding in the epidemiology studies. In fact, Criteria Document makes a major exception to one of its own decision rules by including diesel

particulate matter as a surrogate for ambient particulate matter for immunological effects. Another question I have, if this is the Criteria, is why is the Document organized and presented in the way it is with regard to particle size? Particle size is a major issue with regard to epidemiology studies, or so I understand, and yet it is not a major factor and it's very difficult to tease out of the epidemiology chapter. In particular, I find neither in this chapter nor in Chapter 9 any discussion of the interplay or the relative importance of the size of the particle and the composition of the particulate matter with regard to the potential for effects.

The organization of the chapter, however, suggests another criteria. And that is that the components might have effects that one would expect under possible levels of exposure. If this is the criterion, however, I have other problems. For example, the document says, or seems to, at least to me, to reject bioaerosols as a possible causal agent based on what they say are the low ambient levels of bioaerosols. Yet a very similar

statement is made about metals and as we've heard already today, metals seem to play an important factor in the analysis used of causality and in fact are a major substance used trying to establish mechanisms of action. So I think that in order to decide what we're doing, it would be very useful for at least some of us who are relatively new to at least the ambient particulate matter discussion to know what the decision rules are and what the criteria are so we can see whether these are real inconsistencies or are consistent with what is trying to be accomplished.

But what I'd really like to spend my time on is what some of you know is my favorite topic and that is evaluating complex mixtures of chemicals. Now, the Criteria Document again says that particulate matter itself is a complex mixture but it only discusses mixtures with regard to particles and gases. I find this confusing and I also find very puzzling the fact the Criteria Document doesn't mention nor use any of EPA's own guidance which has been out for, in some cases, more than 15 years on how to evaluate

complex mixtures of chemicals. Nor does there seem to be any use of the more than two decades of experience many of us have had in conjunction with the EPA on evaluating complex and variable mixtures as, for example, the case of hazardous wastes. I find this particularly puzzling because most of EPA's guidance and guidelines goes to the heart of the question; that is, trying to determine what components or characteristics of particulate matter of any complex mixture pose the greatest hazard so that when mixture varies, we can appropriately adjust the potential risk up or down. In contrast, what seems to happen with a lot of the language in the current Criteria Document is that any effect that has been observed in any fraction of particulate matter, any ambient particulate matter or, in some cases, surrogates from particulate matter, seems to be attributed to all particulate matter. This seems contrary to me to what has been done in EPA in other mixtures analyses. On the other hand, when data are available, it seems that the Criteria Document hesitates to draw any conclusions. This may be in part because data

are missing. For example, using metals again, there's no mention, even by incorporation by reference, of other EPA evaluations of metals, nor is there a reference to the vast, not vast, but reasonable toxicological data base on interaction of mixtures.

Finally, I'd be a bad toxicologist if I didn't say I noted the absence of dose-response information. Now dose-response surfaces from mixtures can be complex, but I point out that they are doable, I've given a reference from which this graph is presented. This was a study we did supported by EPA which shows that it is possible. Thank you.

MR. FLAAK: Anyone in the back of the room having difficulty hearing the speakers or is the room acoustics okay? Any problems back there? No? Okay. Thank you.

DR. AMMANN: Thank you for the opportunity to speak to you. I was privileged to be part of a scientific advisory group to the Puget Sound on Clean Air Agency in 1995. The members are listed in my hand-out. Our goal was to develop a PM2.5 goal for the Puget Sound area based on health and the goal we

developed is as listed here. For 24 hours, 25 micrograms per cubic meter, and these are all without exceedance and since they're listed in the hand-out, I won't continue to read the slides. The basis for our evaluation were the studies available at the time which included the health effects studies, ECHO exposures, as well as these studies on mortality. We analyzed both the short-term exposure effects and of air pollution episodes, as well as the long-term effects. We used the strength of association, the consistency among the studies and the coherency among the related effects in our evaluation. The goal that we presented which I showed in the first slide was accepted by the Board in the Puget Sound Clean Air Agency and a stakeholder process was initiated. The process was in response to the recommendation of the Scientific Advisory Group, and it then examined the source categories for particulate matter, devised strategies to reduce their impact and then did an evaluation of the strategies to see whether, in fact, they would reduce the impact. It was found that they did and that if they were, in fact the impact were

reduced, our goal would be achieved.

The implementation is proceeding as we speak. For example, there is a process for diesel reduction which involves ultra-clean, ultra low sulphur fuel. It involves retrofit of diesel fleets and the new technology of diesel engine and there's also a process in place which is looking at reduction of other combustion sources particularly for wood smoke. We find that the new studies support the '96 conclusions in effect on the Criteria Document. There are now more than 70 new time series studies on mortality and we see that, with a few exceptions, they show positive associations and also, if we look at the Brun across study, we look at life shortening, he derived the factor for U.S. men of 1.31 years which is for the exposed population and as EPA calculated out for the Dutch population where Brun effected the same analysis actually means the life shortening of 11.8 years for those who die.

We have concern about the protectiveness of the current 24-hour standard. There is evidence of mortality and morbidity at

levels as low as 20 to 30 micrograms per cubic meter for the 24-hour standard and we have concerns about the form of the standard, the 98th percentile, which essentially allows unregulated 7 days per year. We are not convinced that the annual standard of 15, which is a good standard, makes up for the deficiencies of the 24-hour standard.

These are better on my slides, I'll have to tell you, but... And they're better on the hand-outs that you have. The Puget Sound Clean Air Agency has a camera on Queen Anne Hill pointing at Mt. Rainier that takes pictures morning, noon and night. The mountain is actually here. This is five micrograms per cubic meter. You get a clear view. The monitor is in the view shed so there's a correlation between visibility and the concentration here which are being used in the effort to achieve our goal. The committee then went on to put a legend on the picture which correlated the health effects from the studies. Both of mortality and morbidity health effects. What we found was that on the current PM10 standard of 65 is way above what

the studies are showing and we don't feel it is protective of life or health. We don't know whether there's a threshold for effect. The other major thing we found is that a goal of 25 microgram per cubic meter is achievable. The strategies for voluntary compliance that we used, the stakeholder process and the NAAQS can actually also achieve this. Thank you.

MR. FLAAK: Harriet, thanks for being flexible with our technology and being prepared in many different modes. Thank you. Anne Smith is up now. Jaro Vostal, you're on deck.

DR. SMITH: Hi. My name is Anne Smith and I'm the Vice President of Charles River Associates and I'm going to be focusing my comments on how the material on the epidemiological studies should be interpreted and discussed for uses in the policy deliberations that will be following so it will be appropriately policy relevant. Two specific recommendations on this count for appropriation into the Criteria Document. This is so that the Criteria Document will be able to support a statistically unbiased risk